REVIEWS.

ART. XXII.—*A Treatise on Bright's Disease and Diabetes, with especial reference to Pathology and Therapeutics.* By JAMES TYSON, A.M., M.D., Professor of General Pathology and Morbid Anatomy in the University of Pennsylvania, etc. *Including a Section on Retinitis in Bright's Disease.* By WILLIAM F. NORRIS, A.M., M.D., Clinical Professor of Ophthalmology in the University of Pennsylvania. 8vo. pp. 312. Philadelphia: Lindsay and Blakiston, 1881.

IN his preface the author says, "that, for more than fifteen years, his thoughts, his studies, and his practical work have all been in the direction suggested by these subjects, and that during that time material has passed under his observation which ought at least, if properly used, to have resulted in an experience which should be useful to others." Dr. Tyson has presented his subjects so clearly and concisely, and has selected his material with such sound judgment, that his work cannot fail to be useful to practitioners and students. His aim evidently has been to give in compact form the essential facts, rather than to furnish an exhaustive treatise burdened with discussion of disputed points. The work is provided with two coloured plates, and with thirty-six wood-cuts, which are devoted for the most part to the structure of the normal kidney and to the appearances of casts.

The first chapter contains a concise and excellent description of the *structure of the normal kidney.* The results of the important researches of Heidenhain, Schachowa, and Langhans are given. Klein's divisions of the uriniferous tubule are adopted. If the recent observations of Ribbert are confirmed, it will be necessary to modify the statement as to the existence of nuclei in the capillary walls of the glomeruli. The first chapter also includes some suggestions as to the methods of preparing the kidney for microscopical study, and a brief consideration of the theories regarding the process of secretion of the urine. In respect of the latter the view accepted is that of Bowman as elucidated by the experiments of Heidenhain.

The second chapter is devoted to the *methods of testing for albumen,* and to a consideration of the *sources and of the mechanism of production of albuminuria.* As was to be expected from the author's experience, his directions for detecting albumen in the urine are eminently simple and practical. He justly condemns the careless, but common, habit of designating by percentages the amount of albumen estimated, in the usual way, by observing the proportion between the albuminous precipitate and the bulk of urine tested. While speaking of pus in the urine as a source of albuminuria, the author calls attention to certain cases of vesical disease where with an insignificant quantity of pus there is an altogether disproportionate amount of albumen, although no evidences of renal disease exist. The least satisfactory part of this chapter is that which is taken up with the consideration of the mechanism of production of albuminuria. The author is evidently not cognizant of the experiments of Nussbaum, and of Posner, when he states that the view which holds that albumen transudes through the capillaries of the glomerulus, is based only on theoretical grounds and not on demonstration. The theory adopted that the chief agency in the transudation of albumen in the kidney is increased blood-pressure in the capillaries has lost much of its basis by the experiments and critical analyses of Runeberg, Heidenhain, Cohnheim, Litten, and others, to whom the author does not refer in this connection. Alterations in the capillary walls are admitted as a cause of albuminuria "only in the more acute inflammatory conditions of the kidney."

In the third chapter the varieties, the nature, and the significance of *casts of the uriniferous tubules*, are ably discussed. Pale hyaline casts are considered to be produced by the coagulation of a fibrinous or albuminous exudation from the blood into the tubes. This view, which is by far the most probable one, has received welcome confirmation by the recent experiments of Ribbert, who has, moreover, demonstrated their albuminous nature in opposition to the conclusions of Axel Key and Rovida. Waxy casts, however, Dr. Tyson thinks, are probably produced by fusion and hyaline transformation of desquamated epithelium. He was able to confirm the observations of Bartels, and of Rindfleisch, that these casts exceptionally give the iodine reaction characterizing the amyloid change. He says that he has never found true casts in urine from healthy kidneys, although he is unwilling to deny the possibility of their occurrence under these circumstances. He has repeatedly had opportunity to confirm the fact that casts may remain in the urine for some time after albuminuria has disappeared.

In chapter fourth, after mentioning some of the different *classifications of Bright's disease*, the author gives his own classification, which is as follows: *acute parenchymatous nephritis, chronic parenchymatous nephritis, lardaceous disease, chronic interstitial nephritis, and cyanotic induration.* This is essentially the classification adopted by Bartels, whose authority is followed in the ensuing chapters perhaps more than that of any other writer. In the present state of uncertainty concerning what renal changes shall be included under the name Bright's disease, concerning the pathological anatomy of recognized forms of the disease, and concerning the groups of symptoms which characterize different varieties of the disease, it is easy enough to find fault with this as well as with any other classification ever suggested. The classification adopted by Dr. Tyson is a convenient and useful one, and in its main features is perhaps more generally accepted at present than any other. The propriety of including chronic passive congestion of the kidney under the heading of Bright's disease, is very doubtful. Cyanotic induration of the kidneys is not usually accompanied by important structural changes. We cannot forbear offering certain criticisms concerning the popular classification of Bright's disease adopted by Dr. Tyson. In the first place the whole doctrine of parenchymatous inflammation upon which it is based is not in harmony with the best views as to the nature of the inflammatory process. The swollen and clouded condition of cells designated as parenchymatous inflammation is also, and with more propriety, called parenchymatous degeneration, albuminous or granular degeneration, etc. While it may attend inflammation, it is also met with under circumstances where the inflammatory nature of the change is not to be thought of. We believe also that the most careful investigators are gradually reaching the conclusion that in the majority of cases of so-called parenchymatous nephritis, whether acute or chronic, alterations in the glomeruli and in the interstitial tissue are present which it is wholly gratuitous to assert are unessential or merely secondary to changes in the epithelial cells. So marked is this tendency of recent investigations that Greenfield, in his *Résumé of the Present Knowledge of Renal Pathology* in 1879, regards as the only essentially parenchymatous form of Bright's disease, certain cases of non-scarlatinal acute nephritis which he admits are as yet imperfectly investigated and may hereafter be shown to be associated with other than purely parenchymatous changes. That a simple parenchymatous degeneration may occur in the kidney as well as in other organs cannot be denied, but there seems to be little ground, either from a clinical or from an anatomical point of view, for calling this change Bright's disease. With what propriety can we bring under the head of acute parenchymatous nephritis, those cases of scarlatinal nephritis in which there are marked changes in the glomeruli, and in the interstitial tissue, sometimes with and sometimes without parenchymatous degeneration of the epithelium of the tubes? What place in this scheme of classification shall we assign to Wagner's acute lymphomatous nephritis, or to Ribbert's glomerulo-nephritis with interstitial nephritis, forms of acute Bright's disease which undoubtedly exist?

Each of the forms of Bright's disease included in the above classification is considered in a separate chapter. This part of the book also includes a chapter on *suppurative interstitial nephritis*, and one on *retinitis in Bright's disease*, the latter by Dr. Norris.

For both *acute* and *chronic parenchymatous nephritis*, Dr. Tyson adopts the view so long and strenuously urged by George Johnson, that the primary and essential change is cloudy swelling of the epithelium of the convoluted tubes. Interstitial changes, if found at all, are secondary to the changes in the epithelium, and are not usually present in the early stages of the disease. "They consist in a hypernucleated overgrowth resulting from the proliferation of the connective-tissue corpuscles always present, or from the fixation of wandered-out colourless blood-cells, and are doubtless due to the long-continued hyperæmia." We are glad to see a full account, based upon Langhans's researches, of the alterations in the glomerulus and its capsule. The appearances interpreted by Langhans as referable to proliferation of the capillary nuclei are in reality due, according to Ribbert's recent studies, to the accumulation of white blood corpuscles and of an albuminous substance in the distended capillaries of the glomerulus. Chronic parenchymatous nephritis is divided into two stages: first, a stage of enlargement, or the large white kidney; second, a stage of atrophy, or the fatty and contracting kidney.

The symptoms of parenchymatous nephritis in its two forms are systematically and well described. After describing the symptoms usually produced by acute nephritis, mention is made of those interesting and rare cases of the disease without dropsy, as well as of the temporary absence, even in grave cases, of albuminuria and casts. In chronic parenchymatous nephritis, general dropsy is almost always present, accompanied by anæmia and debility. The urine in the first stage is diminished in quantity, of low specific gravity, contains a large quantity of albumen, and throws down a copious white sediment containing chiefly granular, fatty, and waxy casts, granular débris, free fatty cells, leucocytes, and occasionally red blood corpuscles. The normal constituents, particularly the urea, are diminished. Dr. Tyson's experience is opposed to Bartel's statements, that at the height of the disease the specific gravity is above the normal, and that the quantity of albumen rises with the specific gravity. In the author's experience urines of low specific gravity contain the most albumen. Uræmia is rare in this form of nephritis, partly because, as Bartels had suggested, the physiological sources of urea are diminished by mal-assimilation and diminished tissue-waste, and partly because some of the urea may be stored in the dropsical fluid. The stage of atrophy is to be diagnosed by knowledge of the previous history, by hypertrophy of the left ventricle, by diminution of the dropsy, by increased quantity of urine and lessened amount of albumen. In distinction from the contracted kidney of interstitial nephritis, the quantity of urine is still within the normal, and contains more albumen, and larger, more numerous, and various casts than in the latter disease. Uræmia is still infrequent, but is rather more common than in the stage of enlargement. Retinal changes are less common than in interstitial nephritis. The prognosis is unfavourable as regards recovery, most cases terminating within two years, and sometimes within a few months. Over twenty pages are devoted to the treatment of acute and chronic parenchymatous nephritis. The therapeutical views inculcated are marked by good sense and sound judgment. In acute nephritis, rest, quietude, and warmth are to be maintained. If the urine is scanty, dry or wet cups and warm fomentations to the loins are to be employed. Purgatives are recommended, not only to secure a complemental act of secretion, but also to relieve congestion, and to promote the absorption and prompt action of other remedies. For diaphoretic effects, jaborandi or pilocarpine and warm baths or packs are to be employed. The infusion of digitalis, the salines, and diluent drinks are the best diuretics. Milk is the typical food; animal flesh should be avoided, because it increases the production of urea. Bleeding from the arm is recommended for the relief of uræmic coma and convulsions, if the patient is not too feeble. Excellent results sometimes follow the administration of chloral hydrate in convulsions. He cautions against the use of opium in uræmic convulsions, and believes that he has seen death accelerated by a dose of opium in itself insufficient to produce fatal results. In convalescence from acute Bright's disease, as well as in chronic Bright's disease, tonics should be employed. In chronic Bright's disease, the author has never succeeded in diminishing the quantity of albumen by tannic and gallic acids. He thinks that he has obtained some effect in this direction by the use of ergot. He has observed a diminution in the quantity of albumen under the use of sandalwood-oil, and recommends it for trial in cases of obstinate albuminuria. Fuchsin and rosanilin he has not tested.

Three stages of *lardaceous disease of the kidney* are distinguished: first, a stage of waxy degeneration confined to the glomeruli; second, one in which the degeneration has extended to the vasa recta and other bloodvessels, and possibly to the membranæ propriæ and even to the epithelial cells of the tubes; and third, a stage of atrophy. In the first stage there are no changes in the epithelial cells; in the second these may be fatty; in the third there is "hypernucleated intertubal overgrowth" as the author rather curiously expresses it. This division we believe to be essentially schematic and not based upon pathological observations. It is doubtful whether there is any form of waxy kidney of any clinical importance, where nephritic changes in the epithelial cells and in the interstitial tissue are absent. As regards the causes of this disease, it is to be noted that the author, together with George Johnson, regards chronic albuminuria itself as a cause of waxy degeneration, and thus explains in certain cases the combination of chronic parenchymatous nephritis, with waxy disease of the kidney. In lardaceous renal disease, there is general dropsy, but this is rarely as extreme as in parenchymatous nephritis. The urine is increased in quantity, of low specific gravity, light in colour, loaded with albumen, and contains but few casts, these being chiefly hyaline and waxy; but in addition perhaps fatty casts and free fatty epithelium may be found. Uræmia is extremely rare, as the urea and extractives are eliminated in sufficient amount to prevent this. Of capital importance in diagnosis is the previous history of the patient, and the recognition of coincident waxy enlargement of the liver and spleen, but even under these circumstances unusual difficulties may attend the diagnosis. The author does not emphasize sufficiently the considerable variation in symptoms which may be observed in different cases of waxy kidney. No mention is made of the cases of even extensive waxy disease of the kidneys without albuminuria which have been reported by Litten. Under treatment especial importance is very properly assigned to prophylactic measures.

Under the causes of *interstitial nephritis*, a remarkable example of the influence of heredity in the causation of Bright's disease is mentioned. In this instance, which came under the author's notice, it was determined that no less than eleven members of one family had been affected with the disease. A similar, although less striking, instance is known to the reviewer, and it is believed that more attention should be given to hereditary influence in the etiology of Bright's disease than has hitherto been the case. The influence of alcohol as a cause of Bright's disease, the author thinks, has been over-estimated. Although the parts of the work relating to the causation of the different forms of Bright's disease are reasonably full, yet we believe that their value would have been increased still further by the introduction of some of the results obtained by Bamberger's analysis of over 2000 cases in which a post-mortem examination was made in Vienna. The morbid changes observed in kidneys affected with chronic interstitial nephritis are well described. We can hardly understand, however, why so well known a fact as "the presence of an increased amount of fat surrounding the contracted kidney, even in emaciated subjects," should be mentioned as a recent discovery of Da Costa and Longstreth. Johnson's views as to the nature and cause of the cardio-vascular changes in this form of Bright's disease are, in the main, those adopted by the author. He admits, with Gull and Sutton, also an increase of the outer or fibrous coat of the arteries, but he dismisses very summarily the views of these writers as to the primary nature of the arterio-capillary fibrosis, and as to the cause of the hypertrophy of the heart. For the latter Johnson's "stopcock theory" is accepted. The observations and views of Da Costa and Longstreth relating to lesions of the renal ganglia and the cause of hypertrophy of the heart, are stated without criticism. The author cautions against mistaking senile atrophy of the kidneys for true interstitial nephritis, and thinks that Gull and Sutton have fallen into this error. The clinical history with all its insidious characters, the complications, the diagnosis, the prognosis, and the treatment of chronic interstitial nephritis, are handled with a full appreciation of the important contributions made to our knowledge of this disease within the last few years.

Dr. Norris's chapter on *retinitis in Bright's disease* is to be commended for its conciseness, and at the same time fulness as regards the essential points. It opens with a short historical introduction, and closes with a reference to treatises containing a full discussion of the subject.

The author regrets that he has no contributions to offer concerning the presence of bacteria in *suppurative interstitial nephritis.* This we also regret, for we believe that by proper methods their presence can be demonstrated in the majority, if not in all, of the cases of this disease in the early stages. This is one of the few diseases in which the pathogenetic influence of bacteria is easily demonstrable. The author's view, however, is that, "supposing the observations of Klebs and Ebstein to be correct, the question of the parasitic origin of pyelo-nephritis becomes only a part of that of the parasitic origin of disease generally, which, to say the most, must be considered, *not proven.*" The author propounds the singular question: "How long can obstructed conditions of the ureter and pelvis of the kidney exist before producing suppurative nephritis?" His answer is that after three years, if not before, "the corresponding kidney or kidneys are probably the seat of suppurative nephritis." The obstructed conditions are of course not necessarily ever followed by suppurative nephritis, the former may exist for a long lifetime without the latter.

Cyanotic induration of the kidney is regarded as a form of interstitial nephritis without marked lobulation of the surface, and with less interstitial new-growth than in the cirrhotic kidney. The process is "in the main one of simple hypernutrition resulting from a copious access of pabulum." Inasmuch as the affected kidney receives less than the normal quantum of arterial blood, this explanation cannot be considered a very fortunate one. While there is no doubt that chronic interstitial nephritis may accompany valvular disease of the heart, and doubtless in some cases be a result of the latter, we do not believe that anatomical investigations sustain Dr. Tyson's position that the ordinary cases of cyanotic induration are examples of interstitial nephritis. The symptoms are those usually described as attending cardiac disease with chronic passive congestion of the kidneys.

The last eighty-four pages of the book are devoted to the consideration of diabetes mellitus and diabetes insipidus. Dr. Tyson's account of *diabetes mellitus* is admirable in all respects. He gives fully the different theories as to the pathogenesis of the disease. For the sake of completeness Huppert's theory advocated by Pettenkofer and Voit, might have been added to the chaotic mass. Excepting Ebstein's observations on renal changes, all that is known of the pathological anatomy of diabetes mellitus is presented. Under causation the author notes the rather frequent occurrence of the disease in physicians engaged in laborious practice. Although he has no especially novel views to offer concerning the symptoms and the treatment, his consideration of these subjects is so systematically arranged and so full of practical suggestions that it will be a most useful guide to practitioners. The theories concerning the cause of diabetic coma are briefly mentioned but not criticized. A description of the appearance of the fundus oculi, called by Dr. Heyl intraocular lipæmia, is introduced, and the condition is illustrated by a coloured lithograph in the frontispiece. Dr. Tyson naturally gives the first place to the dietetic and hygienic management of the disease, and here his suggestions will be found to be in accordance with the results of the best experience and physiological experiments. A convenient summary of articles of food suitable for diabetics is introduced. From the medicinal treatment may be selected, the recommendation of ergot as a useful and rationally indicated drug.

Diabetes insipidus is believed by the author to be the result of some irritation, either direct or reflex, of a centre in the medulla oblongata above the diabetic centre, or of the sympathetic ganglia in the abdomen. "The immediate cause is a dilatation, first, of the arterioles, and then of the capillary bloodvessels of the kidney, through whose thinned and stretched walls, the water osmoses readily," although the possibility of an excito-secretory influence exerted upon the gland-cells of the kidney, as suggested by Eckhard, is admitted. As regards prognosis, "recovery is almost as infrequent as death." Among the therapeutical measures which the author mentions are the use of ergot, blisters at the nape of the neck, and the constant galvanic current. In general the supply of water should not be limited.

Dr. Tyson's treatise we believe has been prepared with great care. It is a clear and valuable presentation of the subjects of which it treats.

W. H. W.

Art. XXIII.—*Osteotomy, with an Inquiry into the Ætiology and Pathology of Knock-knee, Bow-leg, and other Osseous Deformities of the Lower Limbs.* By William Macewen, M.D., Surgeon and Lecturer on Clinical Surgery, Glasgow Royal Infirmary. 8vo. pp. xvi., 181. London: J. & A. Churchill, 1880.

This book is worthy of the closest study by surgeons. It deals with a subject in which great advances have been made of late years. It is the work of a gentleman who has made valuable contributions to its literature, of one who evidently writes from an experimental knowledge of the matter.

Dr. Macewen, and it is a great pleasure to an American to be able to speak of so good a surgeon as doctor, writes in a singularly simple and unpretentious way, which makes the reader feel as if he was being talked to, and at once brings him into close sympathy with the writer. Yet the very straightforwardness of the author has apparently prevented his bestowing that care upon his style, by which alone, in the absence of extended practice in writing, perfect clearness of expression can be secured. For this reason, and from the importance of the details of the subject, the book is one not to be lightly skimmed, but rather to be carefully read, and sometimes pondered over, if the teachings of its writer would be thoroughly understood.

The first chapter is devoted to a consideration of the etiology of those curvatures in bone which generally make a resort to osteotomy necessary, and Dr. Macewen does not hesitate to declare that they are traceable to rickets—thinking, as he does, that, from some cases which have occurred within his own observation, he is justified in extending the definition of rachitis to the disease established after the years of childhood.

Our author thinks that while rachitis is not generally hereditary, it is so in some cases, and that, contrary to the received opinion, the disease is sometimes derived from the father. This last fact he thinks is supported